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Cancer in a Factory Using Amosite Asbestos

E D ACHESON, M J GARDNER, P D WINTER AND C BENNETT

Acheson E D (MRC Environmental Epidemiology Unit, Southampton University, Southampton General Hospital, Southampton SO9 4XY, UK), Gardner M J, Winter P D and Bennett C. Cancer in a factory using amosite asbestos. *International Journal of Epidemiology* 1984; 13: 3-10.

The paper describes the mortality experience of 5969 men employed in a factory where insulation board was manufactured using amosite asbestos from 1947 to 1979. 422 (7%) of the men were known to have died by the end of 1980. Among the 4820 men engaged in the manufacture of insulation board a doubling of the risk of lung cancer has occurred (57 deaths observed; 29 deaths expected). An excess is present both in men who entered the factory before and after 1960. Among the 2461 for whom smoking information is available a detectable excess risk is limited to current smokers exposed to higher levels of asbestos. Apart from five deaths from mesothelioma no statistically large or significant excesses of mortality from cancers of other sites have occurred, but further follow-up of the cohort is in progress. Nine deaths from asbestosis have been recorded. The results are discussed in the light of other studies of the effects of exposure to amosite asbestos.

Amosite (the fibrous form of cummingtonite–grunerite) is an amphibole asbestos which has been used in the past in the United Kingdom principally in the manufacture of insulation board and of asbestos cement for pressure piping.[1] Because populations exposed exclusively to amosite and not to other types of asbestos are rare, less is known about the effects of its dust in man than of the effects of chrysotile or crocidolite. This paper reports the occurrence of cancer among the workforce of a factory in London which has used a mixture of commercial amosite and chrysotile but not crocidolite in the manufacture of insulation board. The occurrence of five deaths certified as due to mesothelioma (four pleural and one peritoneal) in this workforce has already been reported.[2]

DESCRIPTION OF THE FACTORY

The present company (Cape Boards, Uxbridge) took over the site from a flint brickworks in 1945 but brickmaking continued on part of the location until 1974. Amosite has been used in the manufacture of insulation board from 1947, and since 1973 has been the only type of asbestos in use. Very short chrysotile fibre (grade 6 or 7) was also used during the period 1946 to 1973 but, apart from during some months in 1946/47, amosite has been the predominant fibre. The amount of chrysotile added varied from nil to about 3% by weight according to market conditions. Crocidolite was used on an experimental basis for five short trials each lasting less than one day in March, April, May and October 1962 and in July 1964. The experimental product was never manufactured on a commercial basis. No record has been found of the use of crocidolite for any other purpose. The proportion of asbestos in the product varied from 20–40%. Lime (25%), silica (25%) in a form of diatomite or ground quartz—and cement (20%) were also used. In the early years some high density insulation block was also manufactured from amosite.

Until a change in the process in 1964 some of the men in the area where the raw materials were handled were exposed to silica as well as to asbestos dust. As a subsidiary operation vermiculite was exfoliated in a small area of the factory for use off site. It was not incorporated in the insulation board. In the early years dust was controlled by centralized exhaust systems for each building which were ineffective by today's standards. The first major improvement was the transfer in 1964 of that part of the process where asbestos was unbagged and opened (the beater floor) to a new building and its enclosure in 1966. In some areas counts of 30 fibres per ml were found when measurements began in the late 1960s but prior to 1964 much dustier conditions were probably encountered notably on the beater floor. Since that period there have been other improvements. A survey of insulation

MRC Environmental Epidemiology Unit, Southampton University, at Southampton General Hospital, Southampton SO9 4XY, UK.

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board manufacturers (which included this factory) by the Health and Safety Executive showed that during the years 1972 to 1978 about 90% of the measurements were less than 2 fibres per ml.[3] During the period 1947 to 1973 when both amosite and chrysotile were used all asbestos workers would have been exposed to both types of fibre. In 1978 the use of asbestos was gradually eliminated from the process and by 1979 its use had been discontinued.

THE WORKFORCE

A register was made of all individuals ever employed at the plant since it was taken over in 1945 up to the end of 1978. This included 59 people who were working in the factory prior to the introduction of asbestos in 1947. Some of these subsequently were exposed to asbestos, and so a distinction is made between dates of first employment and first exposure. Data were abstracted for a total of 6722 people. Table 1 shows the total number of records abstracted, and those excluded (141) because insufficient identification data were available for follow-up purposes. In addition 62 of the men followed were excluded from the analysis because there were insufficient data to determine whether or not they had been exposed to asbestos.

This study describes the mortality up to the end of 1980 of 5969 men for whom the date of first exposure to asbestos and the minimum data necessary for identification in the National Health Service Central Register at Southport were available. 4820 men are known to have been involved in the manufacture of products containing asbestos. Of the remaining 1149 men 650 (57%) worked in the brickworks, 254 (22%) were office workers, and the remaining 245 (21%) were canteen workers, cleaners etc. In the analysis comparisons are made between the mortality of the men involved in the manufacture of asbestos products and the remainder who are referred to as 'other workers'. In view of the fact that the brickworkers and canteen and clerical staff who constituted the latter group worked within the same factory perimeter (but in different buildings) as the asbestos workers we do not exclude the possibility that they were also exposed to asbestos although presumably at a lower level than the asbestos workers. The degree of exposure of this group is described by the use of the term 'background' in Table 6.

TABLE 1 *Number of records abstracted by completeness of data for follow-up purposes.*

Quality of record	Sex	Number of persons	
Satisfactory	Men	6031	
	Women	550	
Unsatisfactory	—	141*	17 unknown sex 101 unknown date of birth 51 unknown date of entry to firm
Total		6722	

* The subtotals add up to a greater number than 141 because on some records more than one item of information was missing.

The workforce expanded and changed in character in a number of ways during the period. A relatively small proportion of the asbestos workers (654—14%) and of the others (236—21%) started work before 1960, ie more than 20 years prior to the completion of the study. Among these early employees the turnover of the labour force was relatively low and about 40% stayed with the firm for more than five years. A major expansion of the workforce took place during the period 1960–69 when no fewer than 3973 men were taken on. After 1960 the turnover was much higher, with over 70% leaving within a year of joining and less than 10% staying for more than five years. The relative importance of the brickworks in the employment of the labour force declined over the period of the study.

The same company managed a large asbestos factory in the East End of London which used crocidolite, amosite and chrysotile and which closed in 1968. According to the evidence of longstanding employees none of the workforce apart from two foremen were transferred to this factory.

All recorded job titles and the dates of transfer from one job to another were abstracted from the personnel records of each man. An industrial hygienist, who had no knowledge of the mortality data, classified the job titles according to five categories of exposure to asbestos dust—heavy, medium, low, casual and background taking into account the nature of the work and the dates during which it was carried out. Asbestos production workers and supervisory staff involved in production were classified as having experienced 'heavy', 'medium', or 'low' exposure according to the nature of their work and the dates during which they were employed. The 'casual' category contained tradesmen such as fitters, carpenters, etc. who were exposed intermittently to high levels of dust.

The classification was built up on the basis of information derived from records and from the evidence of long-serving employees about the history of the processes of production since the factory opened and the various improvements in ventilation

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that had taken place over the years. The classification is largely subjective as no dust measurements were available before 1969. However in the 'heavy' category of exposure dust levels must have been very high because even after the improvements which occurred in the mid-1960s levels as high as 30 fibres per ml were found in some areas. In the lowest (background) category occasional exposures greater than 2 fibres per ml may have occurred. Each job was given a separate assessment of degree of dustiness during three time periods: 1947–1963; 1964–1970 (following the improvements noted earlier); and from 1971–1978 (following the adoption of the 2 fibre per ml hygiene standard). Where a man had more than one job he is classified here as doing the one considered to be the most dusty.

RESULTS

Table 2 shows the distribution by year of birth of the 5969 men included in the mortality analysis. It shows that the cohort is relatively young at present with over 40% of the men being born after 1940. Table 3 shows the number of men who were alive, dead, had left the country or had been lost to follow-up for other reasons on 31 December 1980. The proportions who had died or were lost to follow-up were almost identical in the asbestos workers and others. Taken overall 422 (7%) of the men had died by the end of 1980.

TABLE 2 *Number of asbestos and other workers by calendar period of birth.*

	Asbestos workers		Others	
Year of birth	Number	%	Number	%
Before 1921	591	12	178	15
1921–30	687	14	138	12
1931–40	1406	29	288	25
1941–50	1814	38	444	39
1951–60	322	7	101	9
Total	4820	100	1149	100

TABLE 3 *Number of asbestos and other workers by vital status at the end of the follow-up period.*

	Asbestos workers		Others	
Status at the end of 1980	Number	%	Number	%
Alive	4203	87	993	86
Dead	333	7	89	8
Emigrated	159	3	35	3
No trace	125	3	32	3
Total	4820	100	1149	100

Table 4 shows the observed and expected numbers of deaths and the standardized mortality ratios (SMRs) for all causes combined, for neoplasms, for diseases of the circulatory and respiratory systems, and for injuries and poisoning. The expected numbers of deaths were calculated by the person-years method using national (England and Wales) death rates for men by age and calendar period. Significance tests and confidence limits (CLs) were calculated by the usual methods based on the Poisson distribution. The only significant excesses of mortality are amongst the asbestos workers. There is an excess from neoplasms (SMR 144; 95% CLs 119, 174) and from deaths due to injury and poisoning (SMR 141; 95% CLs 104, 186).

The excess of deaths from injuries and poisoning among the asbestos workers was unexpected and deserves comment. Thirty of the deaths were due to injuries and 18 to poisoning and other external causes. Only one of the 30 deaths from injury was due to an industrial accident but 12 were due to suicide. In contrast, only one of the 11 deaths from injury among the non-exposed was due to suicide. As none of the suicides took place while the men were employed at the factory and asbestos related disease was not mentioned as a contributory cause of death on any of the death certificates it seems unlikely that these deaths were related to overt asbestos-induced disease.

The excess mortality from diseases of the respiratory system is not statistically significant. However, nine of the observed deaths were certified as due to asbestosis, for which the expected number is virtually zero. Seven of these nine deaths were in men who commenced work during 1947–1959, and the other two started in 1960 and 1962. All these deaths occurred more than ten years after first exposure, with periods of employment ranging from 7–24 years except in one man who worked for less than one year in the factory. Removing these nine deaths there is no indication of any excess in other non-malignant respiratory disease. There was one death from pneumoconiosis due to silica in a brickworker among the non-exposed men.

Table 5 shows the mortality by exposure for each site of cancer where one or more deaths were reported. The main finding is that the excess mortality among asbestos workers is virtually limited to cancer of the lung and pleura (SMR = 210; 95% CLs 160, 269). There was a total of 61 deaths in this category, of which 57 were certified as cancer of the lung. The numerical excess of observed over expected deaths due to lung cancer was 28. In interpreting this finding it

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TABLE 4 *Observed deaths, expected deaths and SMRs from all causes and major systems (ICD, 9th Revision) amongst asbestos and other workers.*

Cause of death	Asbestos workers (68922)† Obs	Exp	SMR	Other workers (16609)† Obs	Exp	SMR
Neoplasms (140–239)	109	75.5	144**	24	23.8	101
Circulatory system (390–459)	117	127.8	92	39	41.8	93
Respiratory system (460–519)	38	29.1	131	6	11.3	53
Injury and poisoning (800–999)	48	34.2	141*	11	8.7	127
Other causes	21	32.2	65	9	10.5	86
All causes	333	298.8	111	89	96.0	93

* significant at 5% level.
** significant at 1% level.
† person-years of follow-up.

TABLE 5 *Observed deaths, expected deaths and SMRs from cancer by site amongst asbestos and other workers.*

Cause of death	Asbestos workers Obs	Exp	SMR	Other workers Obs	Exp	SMR
Cancer of:—						
Oesophagus (150)	2	2.0	100	1	0.6	161
Stomach (151)	7	7.5	94	4	2.5	159
Colon (153)	6	4.4	137	2	1.4	142
Rectum (154)	4	3.2	124	0	1.1	0
Pancreas (157)	3	3.1	96	0	1.0	0
Lung (162, 163)Δ	61†	29.1	210**	10	9.4	106
Skin (172, 173)	1	1.0	102	0	0.3	0
Prostate (185)	2	2.1	94	2	0.9	218
Testis (186)	0	1.1	0	1	0.3	377
Bladder (188)	2	2.2	89	1	0.8	124
Other urinary (189)	2	1.4	139	0	0.4	0
Brain etc. (191, 192)	5	3.0	165	0	0.8	0
Hodgkin's disease (201)	1	1.6	64	0	0.4	0
Leukaemia (204–208)	4	2.6	154	0	0.7	0
Other cancers	9††	10.0	90	3	2.9	103
All malignant neoplasms (140–209)	109	74.4	147**	24	23.5	102

† 4 of the 61 deaths were from mesothelioma of the pleura.
†† 1 of the 9 deaths was from mesothelioma of the peritoneum.
* significant at 5% level.
** significant at 1% level.
Δ the SMR for lung cancer for the District in which the factory is situated was 107 during 1968–78.

should be borne in mind that the SMR for lung cancer for men resident in the district in which the factory is situated (107) is close to the national figure for England and Wales used in the computation of the expected numbers. There was no large or significant excess of cancers of all other sites combined, a total of 48 deaths being observed compared to 45.3 expected. When individual sites are considered in none was the SMR significantly raised. The deaths from cancer amongst asbestos workers include four certified as

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pleural mesothelioma (classified with lung cancer) and one as peritoneal mesothelioma which have been described fully in another paper.[2]

No significant excesses of cancer were observed amongst the men who had not been involved in the manufacture of asbestos products.

Lung Cancer Mortality by Dustiness of Job

In view of the fact that dust levels have fallen during the period covered by the study, mortality from lung cancer has been analysed separately for men who entered the workforce between 1947 and 1959, and those who entered between 1960 and 1978. The mortality from lung cancer among asbestos workers during both the former (SMR 268; 95% CLs 178, 388) and the latter period (SMR 177; 95% CLs 122, 249) are significantly raised. As substantial improvements in the conditions of work occurred in 1965 we examined the mortality from lung cancer of asbestos workers who entered the factory after that date but the numbers are too small to support any reliable conclusion. A slight excess of cases (4 observed; 2.8 expected; SMR 143—95% CLs 39, 366) was found amongst those followed for five years or more, but this is not statistically significant.

Table 6 shows the deaths from lung cancer in the whole workforce classified in terms of the most dusty job which they had undertaken, regardless of how long the job lasted. Striking and statistically significant trends are shown with increasing degrees of dustiness both for men who entered in 1947–1959 and during 1960–1978 ($X^2=4.24$, 1 d.f., $P<0.05$; $X^2=9.65$, 1 d.f., $P<0.01$). A significant trend of increasing mortality with increasing dustiness of work was also found for mortality from diseases of the respiratory system (ICD 460–519). No trend remained when the deaths from asbestosis were removed. No relationship was found between mortality from diseases of the circulatory system (ICD 390–459) and dustiness of work.

Duration of Exposure and Lapsed Period

Table 7 shows that there is no trend of increasing lung cancer mortality with increasing duration of employ-

TABLE 6 *Mortality from lung cancer (ICD 162, 163) by degree of maximum exposure and period of entry to exposure (for definitions of estimated exposure categories see text).*

	Period of entry								
	1947–59			1960–78			1947–78		
Highest exposure category	Person years	Deaths	SMR	Person years	Deaths	SMR	Person years	Deaths	SMR
Background	5127	9	165	11481	1	25	16609	10	106
Low/casual	5029	8	186	12888	4	86	17917	12	134
Medium	7871	16	297	39867	25	194	47738	41	225
Heavy	868	4	536	2399	4	351	3267	8	425
Total	18895	37	233	66635	34	150	85531	71	185

TABLE 7 *Mortality from lung cancer (ICD 162, 163) in asbestos workers by length of exposure.*

	Period of entry								
	1947–59			1960–78			1947–78		
Length of exposure* (years)	Person years	Deaths	SMR	Person years	Deaths	SMR	Person years	Deaths	SMR
Under 1	4414	3	159	40080	15	148	44494	18	149
1–4	3454	8	358	9552	12	282	13005	20	308
5–9	1578	6	469	2773	2	103	4351	8	249
10–14	803	4	392	1641	2	131	2443	6	235
15+	3217	5	132	898	2	293	4115	7	156
Unknown	303	2	876	211	0	0	514	2	689
Total	13769	28	268	55154	33	177	68922	61	210

* Unknown length of exposure occurs when the date of leaving the factory is not known.

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ment at the factory among asbestos workers although in men first employed before 1960 there is an increase among the first three categories. There is some evidence that long-term employees have lower mortality (particularly from lung cancer) than those who leave jobs earlier and this may provide a possible explanation for the subsequent decline.[4] Another explanation may be the overlapping between the period of exposure of the employees and the period of follow-up.[5] An analysis of lung cancer deaths which occurred after leaving the factory, and excluding person–years at risk while in employment, showed in fact a slight rise in the SMR with increasing duration of exposure.

There is also no suggestion of a trend of increasing mortality from lung cancer with increasing time since first exposure (lapsed period) in the results in Table 8. In case such a trend had been obscured by the experience of the large number of men who had worked for less than one year, and whose mortality experience may not be related to work at the factory, these 3021 men were excluded. However, this made no difference to the result.

Smoking

Information about smoking habits (current/ex/or non-smoker) began to be recorded in the medical records of employees in 1971. Data were obtained from most of the workforce employed at that time and from those who joined thereafter amounting to 2461 (41%) of the men included in this analysis. Table 9 shows the relationship between dustiness of job and smoking habits. There is a slight increase in the proportion of smokers with increasing dustiness of work. A lower proportion (25%) of smoking histories were recorded among the unexposed than the exposed.

Table 10 shows the relationship between smoking and the highest asbestos exposure category and lung cancer. The excess risk of lung cancer is limited to asbestos workers who were current or ex-smokers. No increased risk of lung cancer is demonstrable among the small number of non-smokers, but the power of the study to detect a relative risk less than sixfold in this group is low. The expected numbers have been calculated on age–sex specific general population rates, without correction for smoking. This means that

TABLE 8 *Mortality from lung cancer amongst asbestos workers by length of follow-up after first exposure and period of entry to exposure.*

Period since first exposure (years)	Period of entry: 1947–59 Person years	Deaths	SMR	1960–78 Person years	Deaths	SMR	1947–78 Person years	Deaths	SMR
0–4	3001	0	0	19899	2	54	22899	2	45
5–9	2886	6	475	17470	11	209	20356	17	260
10–41	2757	4	219	13590	14	223	16346	18	222
15–19	2592	10	419	4115	6	181	6707	16	281
20+	2533	8	191	82	0	0	2615	8	188
Total	13769	28	268	55154	33	177	68922	61	210

TABLE 9 *Number of men for whom smoking data are available and distribution of smoking habits by degree of maximum exposure.*

Highest exposure category	Percentage of: Non-smokers Number	%	Ex-smokers Number	%	Smokers Number	%
Background	57	20	21	7	214	73
Casual	14	19	1	2	57	79
Low	155	22	50	7	506	71
Medium	226	17	39	3	1030	80
Heavy	13	14	2	2	76	84
All exposures	465	19	113	5	1883	77

TABLE 10

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TABLE 10 *Observed and expected deaths from lung cancer by type of smoker and highest exposure category for men with smoking information.*

Highest exposure category	Type of smoker							
	Non		Ex		Current		Ex and Current	
	Obs	Exp	Obs	Exp	Obs	Exp	Obs	Exp
Background	0	0.4	0	0.5	0	2.1	0	2.6
Casual/Low	0	0.5	0	0.5	4	2.0	4	2.4
Medium/Heavy	1	1.1	3	0.6	18	8.4	21	9.0

the expected numbers for non-smokers are overestimated (by a factor of three or more), but since only one lung cancer death occurred in this group there is no evidence of a significant effect due to asbestos. For smokers the expected numbers would be increased by about 30% if based on rates in smokers, which would still indicate a substantial excess of lung cancer among the medium/heavy exposure group. Using methods described, for example by Saracci,[6] it is found that the data are consistent with a greater than additive, although less than multiplicative, interaction between smoking and asbestos. The SMR from lung cancer was similar among men whose smoking status was known (173) and those among whom it was unknown (193). It seems unlikely, therefore, that bias has arisen through the recording of smoking information being influenced by the cause of death.

DISCUSSION

Apart from the occurrence of five deaths due to mesothelioma reported elsewhere,[2] the principal finding of this study is a substantial excess mortality from lung cancer among men exposed principally to commercial amosite, but with a small quantity of chrysotile being used also. The excess is present both in persons who entered work prior to and after 1960, and is related to the degree of dustiness of work. The highest SMR from lung cancer found in any of the larger subgroups of the total cohort was in men who entered prior to 1960 and who had worked in the two categories of job with the highest dust exposures (cases observed 20; expected 6.13; SMR 326; 95% CLs 199, 504). However, caution is necessary in using this figure to compare working conditions with those experienced by men in other factories because of the wide differences in the selection of workers between studies.

We are unable to explain the absence of convincing trends of increasing lung cancer mortality with duration of exposure in the factory or period since first exposure (lapsed period). An increase in lung cancer mortality with increasing duration of work is clearly seen in the studies of Seidman and his co-workers of the amosite workers in Paterson, New Jersey.[7] Like the New Jersey workers, the exposed men in our study showed an excess mortality from lung cancer after a period since first exposure to asbestos of as little as 5–9 years (Table 7). This may be due to the effect of prior tobacco smoking. Although we do not have complete information about previous occupations in the workforce, we consider that previous occupational exposure to asbestos on a large scale was unlikely because of the absence of an asbestos industry in the area of London in which the factory is situated.

In the Paterson factory[7] the increase in lung cancer mortality extended to men who had worked for very short periods eg, less than one month at the plant but this excess did not become apparent until at least 25 years had elapsed since first exposure. In our study the majority of the men who left within a year—ie 3256 men of whom 1837 (56%) stayed less than three months—entered the workforce after 1960 so that the number of these who have experienced an extended lapsed period is relatively small. They experienced a significantly raised lung cancer mortality after a lapsed period of 5–9 years (9 observed; 3.10 expected; SMR 290; 95% CLs 133, 551) but not thereafter.

Another difference between our findings and those of the Paterson workers is that the men in our study, unlike them, have no evidence of excess risks of cancers other than lung cancer and mesothelioma, for example of cancers of the stomach, colon, rectum and pancreas.[8,9] The view that exposure to asbestos may produce malignant tumours other than lung cancer, mesothelioma and perhaps laryngeal cancer is not yet universally accepted. It has been suggested that the excess mortality from, for example, cancers of the stomach and large bowel experienced by the Paterson workers, American insulators and in certain British factories may be due to substances other than asbestos in the working environment.[1,10] Although some such cases may represent misdiagnosed peritoneal mesotheliomas this is unlikely to account for all of them.[10] The processes and materials used in the Paterson and Uxbridge plants were similar, but not identical, and it

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is possible that the existence of an additional unidentified carcinogen in the American factory may explain the difference. Another possibility is that excesses of cancers other than lung cancer due to amosite may disclose themselves in our workforce when the cohort has been studied for a longer period of time and a larger number of deaths have accumulated. In the Paterson workers and in American insulation workers exposed to amosite and chrysotile the excess risks of alimentary tract cancer are smaller than for lung cancer.[11]

In our material slightly increased relative risks are apparent after a lapsed period of more than 15 years for deaths from stomach cancer (4 observed; 2.39 expected; SMR 167, CLs 46, 428) and cancer of the colon (2 observed; 1.46 expected; SMR 137, CLs 17, 494) but neither increase is statistically significant.

As well as the large excess of lung cancer deaths in this factory, there were nine deaths ascribed to asbestosis among the men. These cases came from among men who had worked in the factory for longer than average periods of time and had higher levels of exposure. The relative size of the number of asbestosis deaths to the excess number of lung cancers (about 1 in 3) is similar to that found in the American amosite factory workers.[7]

The amosite used in this factory and at Paterson, New Jersey, is thought to have come from the same mine at Penge in the Transvaal, South Africa. Although the number of mesotheliomas reported from Penge has been small[1] no formal follow-up of the workforce has been carried out and nothing is known of their risk of other types of cancer.

Little is known about the effect of stopping smoking on the risk of lung cancer in men who have worked with asbestos. In our material the SMR from lung cancer in men who had been exposed to asbestos and were current smokers was 213 (22 observed; 10.3 expected) as compared with 273 (3 observed; 1.1 expected) in ex-smokers but the number of ex-smokers is too small for a reliable inference to be made. In view of the known reduction of risk of lung cancer which follows cessation of smoking in smokers in general it seems prudent to follow Selikoff[12] and his colleagues and take every possible step to advise current and past asbestos workers to stop smoking.

A study of the fibre burden in the lung tissue of the men who died from lung cancer and mesothelioma and in men dying of other conditions is planned.

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